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# Lactation Failure in Swine

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Lactation failure in swine is a rather common disease entity that has perplexed swine producers and veterinarians for generations. While the disease has been studied extensively for years proper prevention and treatment methods have been elusive. The most common name for the disease is the mastitis-metritis-agalactia (MMA) complex. As will be discussed in this review, metritis is rarely a part of the disease. It has been suggested that a more descriptive name for the disease complex is the periparturient hypogalactia syndrome.<sup>1</sup>

Reports indicate that the overall incidence of the disease in the United States is quite variable. A survey in Missouri in 1973 indicated that 13% of the farrowings were affected by the MMA syndrome. In 1972, Leman et al reported that 18% of total preweaning pig mortality in Illinois swine herds was caused by starvation probably as a result of lactation failure. In 1979, Anderson reported that up to 50% of the total preweaning pig mortality was related to MMA. However, recent surveys conducted by the National Pork Producers Council have indicated less impact of the disease when compared to other disease problems. From 1978 to 1982, MMA fell in rank in importance from second to fourth when ranked by pork producers in the United States.<sup>2</sup>

This article will discuss the clinical signs, diagnosis, and the etiologies of PHS. Special emphasis will be placed on the prevention and treatment of the disease.

## Clinical Signs

The sow or gilt that develops the agalactia syndrome will typically have normal milk production

for 12 to 24 hours postpartum. This is followed by partial to complete agalactia. Some difficulty may arise in detecting agalactia postparturiently because many sows will not let down milk except during a 10 to 20 second period of time during the nursing cycle. The condition is most often characterized by hypogalactia that lasts 24 to 96 hours rather than by mastitis, metritis, or agalactia.<sup>3</sup> Vaginal discharge, or lochia, is common in postpartum sows. Lochia generally does not correlate with agalactia. Some have assumed that discharges imply reproductive tract infection. Although some sows do have uterine infection, relatively few have metritis.<sup>4</sup>

Diagnosis of the disease can sometimes be difficult but careful observation of the piglets should give some clues. During the first 24 hours piglets alternately sleep and nurse at regular intervals. During the next 24-48 hours a more consistent pattern of regular nursing at 40-60 minute intervals develops.<sup>5</sup> The behavior of the piglet normally progresses through five phases: (1) jostling for position, (2) nosing the udder, (3) slow sucking, (4) rapid sucking, and (5) final slow sucking and nosing. The sow's pattern of vocalization related to these phases is consistent. The rate of grunting varied little during phases 1 and 2, but increased substantially at about the beginning of phase 3, and then declined. The time when milk could be expressed manually from the teats was found to correspond to phase 4. In a number of nursings milk flow was observed to fail, and these nursings lacked both the period of rapid grunting and phase 4.<sup>6</sup> Expressing milk during the lactation cycle may be helpful. However, in normal sows it has been shown that 27.4% of nursings failed.<sup>7</sup>

It is important to understand the normal lactation pattern of the sow in order to more adequately diagnose the disease. The producer will normally know something is wrong when pigs are born normally and look healthy but then stop growing and several die without any overt signs of disease. Hypoglycemic pigs will show weakness and other

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nervous signs and will often pile-up in order to maintain body temperature. Close examination of the pigs will show them to be unthrifty and gaunted. Since these piglets are very weak they have an increased chance of getting a secondary bacterial infection — most often scours, and they are also more apt to get crushed by the sow.

The examination should continue with the sow. The sow may show no signs of illness. Sows affected with agalactia commonly have an increased rectal temperature, increased respiratory rate, and anorexia. The newborn pigs will also have decreased weight gains. Careful examination should be made of the entire mammary gland since one infected gland could be causing the problem. Necropsy examinations of sows with so-called MMA have shown over 80% to have gross or microscopic lesions of mastitis.<sup>9</sup> It is also important to observe the premises and obtain an adequate history from the producer on his management practices.

### Etiology

Over 30 factors have been incriminated in lactation failure in sows. Many non-infectious causes have been described including: udder and teat abnormalities, toxemia, hypocalcemia and ketosis, chronic ergotism, stress, feeding of alfalfa meal, changing to finely ground feed, omitting bulk from the ration, overfeeding during gestation (especially the last week), underfeeding, vitamin E or selenium deficiency, feeding of excessive protein, imbalanced ration, aflatoxins, zearalenone, prolonged gestation, administration of intestinal antiperistaltic agents, and heredity. Diseases such as TGE, pseudorabies, and erysipelas may also be involved in some cases.<sup>1,8,9</sup>

There is a plethora of agents that can cause lactation failure. Often there may be more than one factor involved, and it is difficult to determine what the primary cause may be. It would be very time consuming and economically impractical to rule-out all possible factors in each case. It is important to look for the most likely cause first, and evidence confirms that coliforms are the most significant bacteria in mastitis of the sow, and mastitis is identified in a high percentage of hypogalactic sows.<sup>10</sup> A question that needs to be answered is why the coliforms set-up a focus of infection in the first place. Possibly, some of the aforementioned factors may be involved more often than is realized. Bacteriological examination of mastitic glands reveals a high incidence of *E. coli* and *Klebsiella pneumoniae* infection. While numerous *Streptococcus spp.* and *Staphylococcus spp.* have been isolated, these bacteria usually are not associated with

pathologic changes in the mammary glands. Mycoplasmas have been incriminated but have not been shown to cause PHS.<sup>1</sup>

Toxic agalactia caused by toxin producing bacteria is the most important cause of agalactia (88.6%).<sup>11</sup> Smith and Wagner at Illinois in 1984 demonstrated that sows are more susceptible to endotoxin induced suppression of milk production at 2 days post partum than at 6 days post partum.<sup>12</sup> This correlates with the early postpartum lactation failure that is most often seen. If a sow or gilt is found that has mastitis in one or more glands it would be reasonable to assume that the mammary gland is the focus of toxin production.

Other sources of a bacterial focus of toxin production have been studied. One study's data suggests that the mammary gland and ileum are the most likely sources of endotoxin in cases of lactation failure in sows. This study also showed data to support the concept that uterine involvement in pathogenesis of the disease is of minor importance.<sup>13</sup> Another study concluded that *E. coli* endotoxin is not readily absorbed from the small intestine, but this study also mentioned that constipation is often listed as a sign of lactation failure. The constipation may cause a higher than normal level of bacteria in the small intestine. Stress or changes in feeding program around parturition may alter the barrier function of the gut epithelium such that endotoxin from the intestinal flora may enter the systemic circulation.<sup>8</sup> However, the jejunum, ileum, and cecum are not likely sites of endotoxin absorption.<sup>14</sup>

Endotoxin can be readily absorbed from the uterus<sup>15</sup>; however examination of the uterus in sows and gilts demonstrating lactation failure is generally non-revealing. Bacteria may be isolated from the uterus, but histologically there is usually no evidence of infection.<sup>3</sup>

The mechanism of action of endotoxin is not completely understood. Endotoxins are believed to be absorbed systemically and may alter the cardiovascular system which decreases mammary perfusion and hence milk production. Endotoxins also cause generalized signs of fever, lassitude, constipation, and other signs by the sow. The sow requires prolactin for normal lactation and low serum and adenohipophyseal concentrations of prolactin in agalactic sows may be an important aspect of the condition.<sup>16</sup> Wagner et al also found decreased prolactin concentrations in blood after postfarrowing administration of endotoxin.<sup>13</sup> It is not known whether endotoxin alters prolactin secretion directly at the level of the adenohipophysis or indirectly through alteration of hypothalamic prolactin releas-

ing inhibiting factors. One study concluded that some, or all, of the effects of endotoxin on prolactin release is mediated directly at the pituitary level. It was not concluded whether the effects were attributable to endotoxins functioning as an antagonist of the prolactin releasing factors or receptors, as a prolactin inhibitory factor agonist, or exerting some other direct suppressive effect on the lactotrophs.<sup>14</sup>

### Control

1. Cleanliness must be stressed in order to minimize teat exposure to coliforms. Sanitation also aids control of baby pig scours.

2. Stress should be minimized in the farrowing house. Epinephrine released when animals are stressed will block the action of oxytocin at the myometrial level.<sup>3</sup> A good recommendation is to have the producer place a radio in the farrowing house and play it at all times in order to minimize disturbance caused by entry of the herdsman into the room.

3. Selection of replacement gilts is important. Gilts should be selected that have at least 12 functional nipples that are evenly spaced. It is preferred that replacement gilts be selected from sows with high indices for maternal ability (i.e. 21 day litter weight).

4. Sows should be kept off feed or fed little during the first 24 hours post partum. Sows should be brought onto full feed gradually during the first week post partum and then fed 4 pounds plus ½ pound per pig nursing.

5. A vaccination program should be instituted in order to reduce coliform scours. Even though the strains of *E. coli* incorporated in vaccines used for control of coliform scours (i.e. K88, K99, and K987p) are not the culprits in lactation failure, producers and veterinarians have reported informally that these vaccinations do aid in the prevention of lactation failure. Evidently, some mechanism of partial cross protection develops which is not understood. Experimental work with autogenous bacterins has given disappointing results. Auto-genous bacterins have been helpful in some herds<sup>5</sup> and useless in others.<sup>5</sup>

6. Feed should be analyzed periodically to ensure that all sows are receiving the proper amount of nutrients daily. In one study Vitamin E and Selenium were supplemented for three to four months to prevent growing pig losses. During this time period the MMA problem decreased and eventually ceased to exist.<sup>17</sup>

7. F series prostaglandins have been shown to reduce incidence of agalactia. Reports have shown

PGF2alpha to be highly effective for induction of parturition in swine at 111 to 113 days gestation. It is also effective at earlier stages, but litter survival is diminished.<sup>18</sup> Duration of birth is shorter, number of pigs alive is greater, and there is a lower incidence of agalactia if parturition is induced. Prostaglandin followed 15-25 hours later with 20-30 IU of oxytocin consolidates the farrowing and improves piglet survival; this regimen facilitates closer observation of farrowing by herdsman.

8. Bulk such as oats or beet pulp should be used in the ration to decrease constipation. This may reduce problems caused by overgrowth of endotoxin producing bacteria in the intestine.

### Treatment

A complete history and examination should be made of all animals in the farrowing house. The producer should be quizzed on his feeding program and management practices. The piglets must be examined to determine if hypoglycemia is the only problem. If secondary infections are present they must also be addressed.

1. Oxytocin has been the standard treatment. 20-40 units can be given every 3 to 4 hours to facilitate milk letdown. Although the dose level of oxytocin necessary to obtain desired responses in agalactic sows has not been determined, indications are that it should be larger than 20 units. The largest dose recommended by the manufacturer for milk letdown is 20 units.<sup>19</sup> Responses to exogenous oxytocin in agalactic sows appears to be less than in normal sows. The major drawback of this technique is the amount of time that must be spent giving injections, and it must be remembered that stress can cause agalactia or hypogalactia, and giving many injections is definitely going to stress an animal. It is unlikely that repeated injections of exogenous oxytocin induces antibody formation.<sup>20</sup> Agalactia due to failure of milk letdown is most common in first litter gilts and is characterized by the inability of the gilt to feed the pigs. These gilts respond very well to oxytocin.<sup>3</sup>

2. Antibiotics should be used if bacterial infection is involved. Antibiotic choice should be made using past history on the farm if possible. If this information is not available, aminoglycoside or trimetoprim-sulfa combinations are most likely to be successful.<sup>5</sup>

3. Administration of glucocorticoids are believed to be effective for PHS in the field. However, there is no clear evidence that they have a beneficial effect. Glucocorticoids are normally elevated during the periparturient period and cortisol is even elevated further in most affected animals.<sup>1</sup> However, cor-

ticosteroids are anti-inflammatory and synthetic corticosteroids are bound less by globulin than are natural corticosteroids.<sup>3</sup> Therefore, there is no clear answer regarding benefits of their use.

4. Tranquilizers can be used to calm restless gilts or sows.<sup>3</sup>

5. If the sows are anorexic and it is believed this and not a secondary sign could be leading to the problem, B-vitamins could be used as an appetite stimulant. The feed could also be changed. However, anorexia is unlikely to be primary problem.

6. Treatment with 2.2 mg/kg of flunixin meglumine (repeated in 12 hours in severe cases) reduces mammary edema and anorexia and improves the piglets' gain.<sup>1</sup> This treatment is not approved in the United States.

7. Dipyrone can be used in febrile sows. This is not approved in the United States.<sup>3</sup>

8. High ceiling diuretics have been used using 1 dose at 12 hours after parturition. This is non-scientifically believed to decrease the amount of milk the animal produces the first few days so that the piglets are more able to completely milk out the sow. This treatment is controversial and is also not approved.

8. Commercial milk replacers can be used. It is best to leave the piglets on the sow and supplement their diets until the sow begins to produce milk normally again.

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